Carotid Sinus Nerve Stimulation in the Treatment of Angina Pectoris and Supraventricular Tachycardia

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DR. E. BRAUNWALD:* The carotid sinus is a dilatation of the internal carotid artery near its origin at the bifurcation of the common carotid artery. This area of the vessel is richly supplied by nerve receptors which lead into the carotid sinus nerve; the latter in turn joins the ninth cranial nerve, the glossopharyngeal, which leads to vasomotor centers in the medulla. Since the classic work of Hering in 1923 it has been known that the carotid sinuses play a critical role in the regulation of arterial pressure. Stimulation of the pressure receptors (baroreceptors) in the carotid sinuses and aortic arch results in reflex arteriolar dilation and reduction of heart rate and myocardial contractility, this as a consequence of a reduction in the frequency of sympathetic efferent impulses and an increase in the frequency of vagal impulses.[1] The opposite changes occur when the pressure acting upon these receptors is reduced. For this reason, the sensory nerves from the baroreceptors are called buffer nerves; in normal circumstances they are continuously active in the regulation of arterial pressure, heart rate and myocardial contractility.

It is well known that profound circulatory changes can be induced by carotid sinus nerve stimulation. For many years manual stimulation of the carotid sinuses has been commonly used for interrupting supraventricular tachycardia. More recently, Schwartz[2,3] and his associates as well as other investigators[4-7] have implanted electrodes on the carotid sinus nerves of patients with hypertension which was unresponsive to conventional treatment, and by stimulating the nerves continuously have reduced the arterial pressure.

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The objective of this conference is to review our experience with intermittent electrical stimulation of the carotid sinus nerves in the management of intractable angina pectoris and supraventricular tachycardia. Our interest in the use of this approach for the treatment of angina arose from a series of investigations on the determinants of myocardial oxygen consumption. These investigations were recently reviewed elsewhere[8]; briefly, however, they demonstrated that the oxygen demands of the heart are not simply a function of the external work of the heart, that is, the product of arterial pressure and cardiac output. Rather, the development by the ventricles of pressure (more strictly speaking, tension) has a relatively high oxygen cost, while the cardiac output is a far less important determinant of myocardial oxygen consumption. In addition to tension, the level of myocardial contractility, that is, the inotropic or contractile state of the myocardium, is a second important determinant of the heart's oxygen needs. A third factor, of course, is that the heart's oxygen consumption is a function of cardiac frequency—the number of times the heart contracts per unit of time. A number of other physiologic variables have been studied, but none even approach in importance the three factors mentioned above—tension, contractility and heart rate—in the control of myocardial oxygen consumption.

It is generally appreciated that angina pectoris results from an imbalance between the heart's oxygen needs and the oxygen supply. In the large majority of patients this imbalance results from a defect in the delivery of oxygen to the myocardium as a consequence of obstruction in the coronary vascular bed secondary to atherosclerosis. In other patients, however, this imbalance results to a significant extent from increased myocardial oxygen demands, as occurs in aortic stenosis, thyrotoxicosis and tachycardia. Ideal therapy for angina pectoris would be to restore the balance between supply and demand in as physiologic a manner as possible—by increasing oxygen delivery when it is limited, and by reducing oxygen demands when these are excessive. In practice, however, this has been difficult to achieve, particularly in patients with diffuse, severe atherosclerosis.

With this understanding of the determinants of myocardial oxygen consumption in mind, we at first sought to relieve angina pectoris by reducing myocardial oxygen consumption through stimulation of the vagus nerve. In collaboration with Drs. Gerald Glick and Andrew Wechsler, we placed radiofrequency stimulators on the right vagus nerve of a group of dogs and, after they recovered from the procedure, observed that we could reduce their heart rates to almost any desired level when they were studied under general anesthesia. However, when the dogs were conscious, electrical stimulation of the vagus nerve always produced serious side effects—coughing, salivating, vomiting—apparently because of activation of fibers in addition to efferent cardiac parasympathetics. We then directed our efforts to electrical stimulation of the carotid sinus nerves, because, as indicated above, activation of these nerves results in reflex reductions of heart rate, arterial pressure and myocardial contractility, the three prime determinants of myocardial oxygen consumption. The experimental results were far more promising and we then studied a group of dogs for periods up to one year and observed that they tolerated electrical stimulation of the carotid sinus nerves without significant deleterious side effects. The observation that manual stimulation of the carotid sinuses can abolish attacks of angina[9] and that in fact Lown and Levine[10] have proposed this as a diagnostic test for angina encouraged us to apply this method clinically in patients with angina pectoris.

Before summarizing the results of carotid sinus nerve stimulation in patients with angina pectoris, Dr. Vatner will describe studies currently in progress in our laboratory on the hemodynamic changes resulting from brief periods of electrical stimulation of the carotid sinus nerves in dogs.

Hemodynamic Changes

DR. STEPHEN F. VATNER:* Our interest in studying the effects of carotid sinus nerve stimulation in the conscious animal arose from the fact that the carotid sinus reflex is of preeminent importance in the reflex control of arterial pressure and heart rate. Although numerous investigations into the effects of carotid sinus nerve stimulation have been carried out in anesthetized animals,[1] little information on the circulatory effects of stimulating these nerves is available in intact unanesthetized animals. General anesthesia has profound influence on circulatory regulation by the carotid sinus

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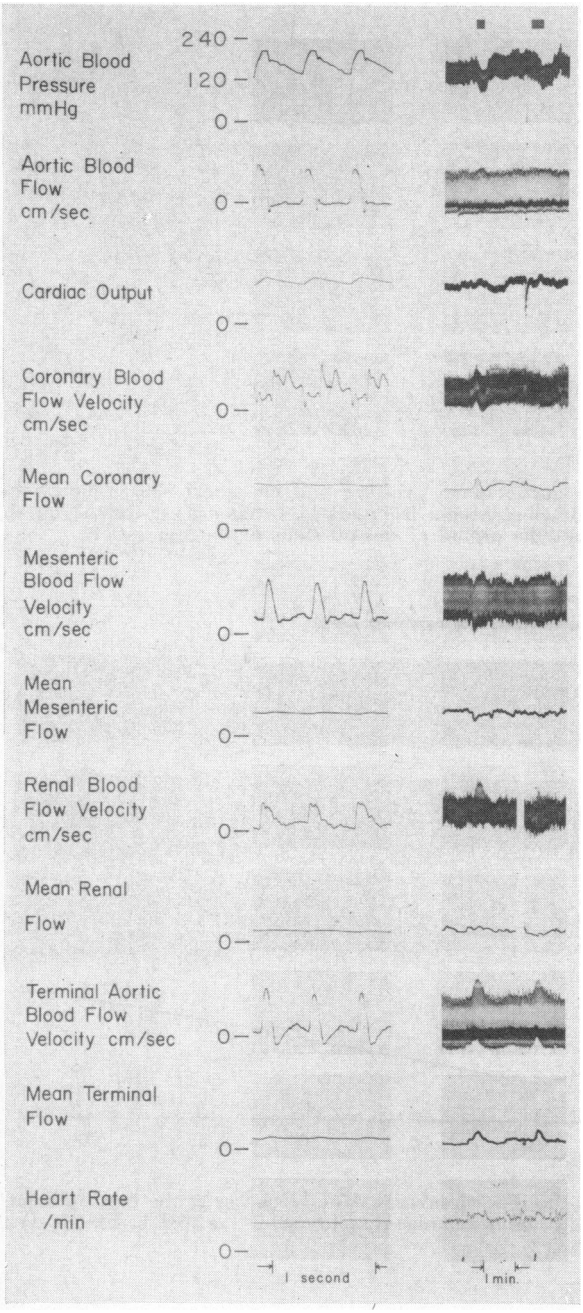

Figure 1.—The effects of carotid sinus nerve stimulation on aortic blood pressure, ascending aortic blood flow, left circumflex coronary blood flow, mesenteric blood flow, renal blood flow, terminal aortic blood flow and heart rate. The periods of stimulation are presented by the black bars at the top. Left panel: Tracing at rapid paper speed. Right panel: Tracing at slow paper speed. All blood flows are shown as instantaneous velocity and mean flow.

nerves.[11] Therefore, we wished to examine the effects of carotid sinus nerve stimulation in healthy animals not under the influence of anesthesia. In view of the increasing clinical applications of carotid sinus nerve stimulation in patients with angina and hypertension, as described by Dr. Braunwald, this investigation in conscious dogs was considered to be particularly important since it might provide information about the carotid sinus reflex which cannot be readily obtained in man. In studies carried out in collaboration with Mr. Dean Franklin and Drs. R. Van Citters and E. Braunwald[11-13] we determined the effects of carotid sinus nerve stimulation on the responses of arterial blood pressure, heart rate, cardiac output and blood flow distribution in the iliac, renal, mesenteric and left circumflex coronary beds.

With the animals under sodium pentobarbital anesthesia, pulsed ultrasonic or Doppler ultrasonic flow probes were placed on the ascending aorta and the left circumflex coronary artery, as well as on the mesenteric, renal and iliac arteries, and miniature solid state pressure gauges were placed in the central aorta. Electrodes were placed on both carotid sinus nerves. One to four weeks after recovery from the operation, with the animals apparently well, experiments were performed in 12 dogs and two baboons. A radiofrequency pacemaker* identical to that used in the clinical studies was used to stimulate the nerves for 30-second periods. The experiments in which the pulsed ultrasonic flowmeter was used were conducted in the laboratory; the battery-operated Doppler ultrasonic flowmeter could be placed in saddle bags so that flow and pressure could be telemetered without the animals being tethered (Figure 1).

Carotid sinus nerve stimulation resulted in an initial fall in aortic pressure, the decrease averaging 20 to 35 percent of resting control levels. However, pressure began to return to control levels even while stimulation was continued and reached control levels shortly after stimulation ceased. Surprisingly, heart rate decreased only slightly, by 12 percent of control, during the initial phase of stimulation and returned to control levels after only about 10 seconds of stimulation, while arterial pressure continued to fall. In some animals heart rate actually increased to above control levels with continuation of stimulation for periods exceeding 20 seconds. After an initial decrease of approximately 10 percent, cardiac output returned to control levels during carotid sinus nerve stimulation. Thus, since arterial pressure decreased significantly, calculated peripheral vascular resistance declined.

*Manufactured by Medtronic, Inc., Minneapolis.

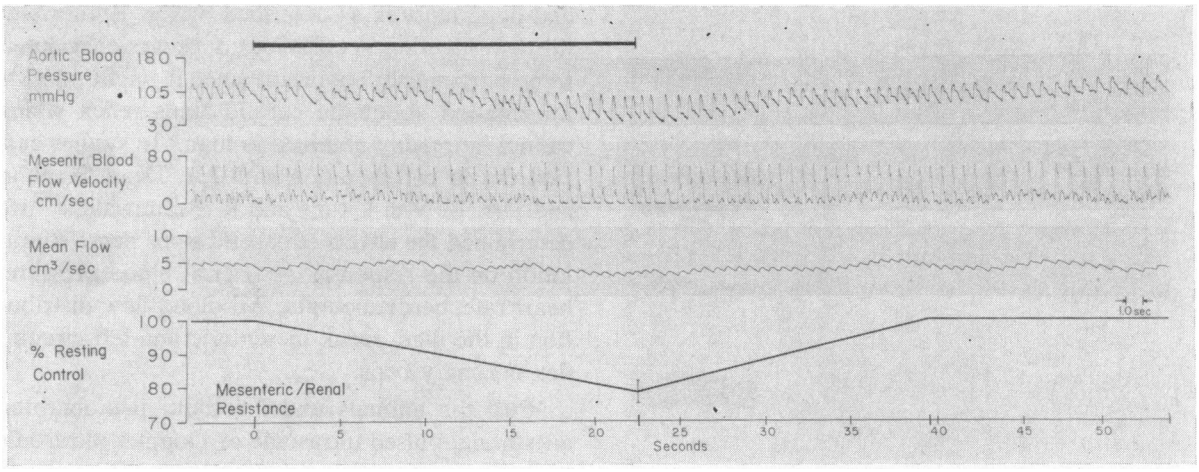

Figure 2.—The effects of carotid sinus nerve stimulation on aortic blood pressure and mesenteric blood flow. The graph at the bottom illustrates the average decreases in calculated resistance from control levels in both the mesenteric and renal beds in six dogs. The black bar at the top represents the period of carotid sinus nerve stimulation.

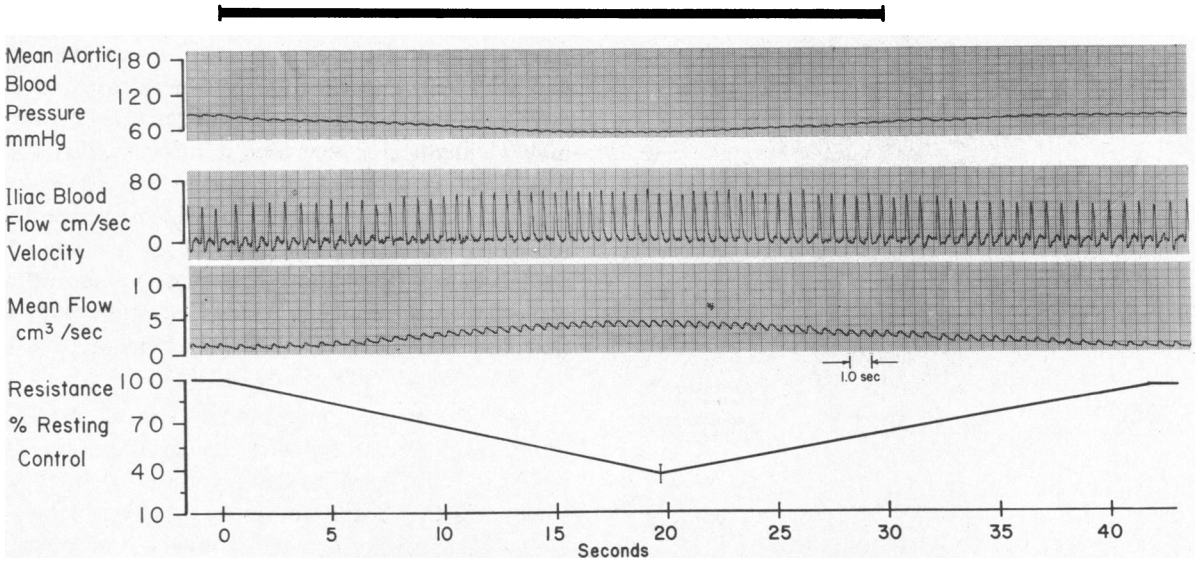

Figure 3.—The effects of carotid sinus nerve stimulation on the iliac circulation. The black bar at the top represents the 30-second period of stimulation. The average maximum decrease in calculated resistance in the iliac bed in six dogs from central levels is demonstrated by the graph at the bottom.

The mesenteric and renal beds responded in a similar manner (Figure 2); in both beds flow decreased during carotid sinus nerve stimulation, but not as much as pressure, resulting in a net decline in calculated vascular resistance in these two beds by approximately 20 percent of control. In contrast to the other beds which we studied, flow to the hind limbs uniformly increased during carotid sinus stimulation, sometimes up to four times the control level, despite the decrease in arterial pressure (Figure 3). Thus, far greater dilatation occurred in the iliac than in any of the other vascular beds, amounting to a 60 percent to 70 percent decrease in calculated vascular resistance.

We anticipated that carotid sinus nerve stimulation would cause no change or perhaps only a slight increase in coronary vascular resistance, since carotid sinus nerve stimulation reduces the metabolic requirements of the heart by lowering arterial pressure, heart rate and contractility, and

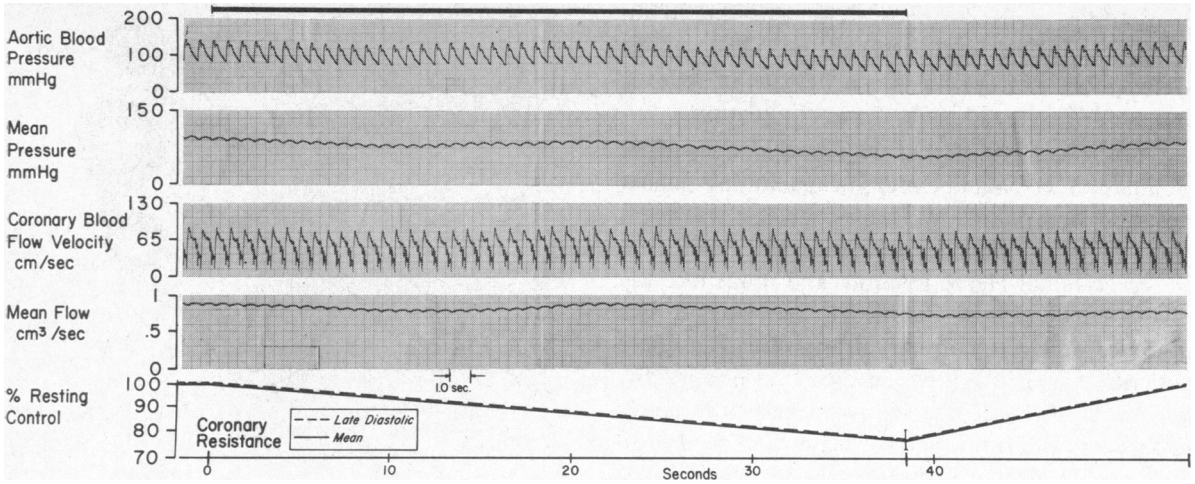

Figure 4.—The effects of carotid sinus nerve stimulation on aortic pressure and left circumflex coronary artery blood flow. The graph at the bottom represents the average maximum decrease in calculated mean and late diastolic coronary resistances in six dogs. The black bar at the top represents the period of nerve stimulation.

since coronary vascular resistance is determined largely by and is inversely related to myocardial oxygen consumption. We were therefore somewhat surprised to find that coronary blood flow remained constant, decreased only slightly, or in some experiments actually increased, and calculated coronary vascular resistance declined by 20 to 25 percent (Figure 4). Subsequent studies in dogs treated with various autonomic blocking agents—atropine, propranolol and guanethidine—revealed that the reduction in coronary vascular resistance induced by carotid sinus nerve stimulation was mediated primarily by a reduction of sympathetic vasoconstrictor impulses to the coronary bed.

Thus, we have found that carotid sinus nerve stimulation in conscious animals produces an expected decrease in arterial pressure, surprisingly little bradycardia, and significant dilatation in all beds studied. The greatest reductions in calculated resistance occurred in the muscular circulation, where flow actually increased while arterial pressure decreased. Of greatest interest and potential clinical significance is the finding that carotid sinus nerve stimulation causes a decrease in calculated coronary vascular resistance. The fall in vascular resistance in all beds appears to be due to release of sympathetic constrictor tone, while the bradycardia is due more to vagal stimulation than to decreased sympathetic activation.

DR. E. BRAUNWALD: Before the performance of the experiments described by Dr. Vatner, it was our impression that the relief of angina resulting from carotid sinus nerve stimulation resulted entirely from a reduction of myocardial oxygen requirements, and the creation therefore of a more favorable relation between myocardial oxygen supply and requirements. Now, we must also consider the possibility that coronary vasodilatation occurs. The observations in conscious dogs just described also complement those which we made in patients together with Dr. Stephen E. Epstein and others.[14] The effects of carotid sinus nerve stimulation were studied at rest and during a mild level of supine bicycle exercise in seven patients in whom stimulators had been implanted for the treatment of angina pectoris. At rest, carotid sinus nerve stimulation produced a fall in mean arterial pressure averaging 23 percent, an 8 percent decrease in cardiac output, and a 9 percent decline in heart rate. Total peripheral resistance fell by 14 percent and forearm vascular resistance by 16 percent. During exercise with carotid sinus nerve stimulation, mean arterial pressure fell by 16 percent but, interestingly, no significant change occurred in the cardiac output, and the decreases in heart rate were very small. Thus, the fall in arterial pressure could be attributed to a reflexly induced decrease in peripheral vascular resistance. No changes in venous tone, central venous pressure, or the maximum transverse end-diastolic diameter of the heart were produced by stimulation, either at rest or during exercise. Thus, at rest, carotid sinus nerve stimulation reduces mean arterial pressure by reflexly decreasing both vascular resistance and car-

diac output, while during exercise, the diminution in cardiac output no longer occurs. The venous system does not appear to participate in reflexes activated by carotid sinus nerve stimulation.

Our clinical experiences with carotid sinus nerve stimulation in patients with incapacitating angina began in June 1967. We have used this treatment in a total of 22 patients with angina pectoris, first at the National Heart Institute and more recently at the University of California, San Diego. The presence of severe coronary artery disease in these patients was proved by coronary arteriography, and in all of these patients was found to be unresponsive to intensive medical management. I shall now ask Dr. Nina S. Braunwald to describe the device used and the operative technique employed.

Operative Implantation of Electrodes

DR. N. S. BRAUNWALD:* The carotid sinus nerves are stimulated bilaterally by bipolar platinum electrodes connected by stainless-steel wires to a receiving unit implanted subcutaneously in the anterior chest wall. The transmitting unit is worn externally and generates 20 to 80 radiofrequency pulses/sec., 0.3 msec in duration and 0.5 to 8 volts in amplitude. The pulses from the signal generator are transmitted by an induction coil that is placed on the skin directly overlying the implanted receiving unit. The patient activates the transmitting unit at will by an on-off switch (Figure 5).

The anesthetic and surgical techniques have been described in detail elsewhere.[15,16] Briefly, however, all drugs are discontinued one to two days before operation. Following premedication, the patients are taken to the operating room where an intra-arterial needle is introduced into the radial artery for constant monitoring of arterial pressure. The heart rate and electrocardiogram are also monitored continuously and any fluctuations are treated immediately with appropriate drug therapy. Following induction of general anesthesia, a transverse incision is made below the clavicle on the anterior chest wall and a small pocket is developed to permit implantation of the receiver. Two transverse incisions are then made below the mandible at the level of the hyoid bone, the carotid sheath is entered and the carotid bulb identified. Umbilical tapes are placed about the internal and external carotid arteries. The carotid sinus nerve

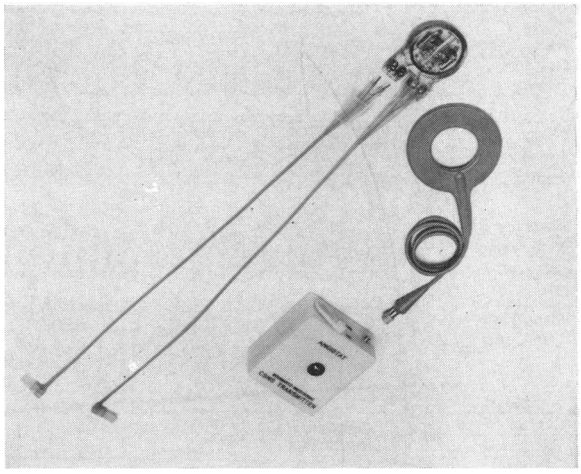

Figure 5.—The radiofrequency stimulator used in the treatment of angina pectoris. The transmitter and antenna are worn externally and are shown at the right. The implanted receiver unit is seen at the top.

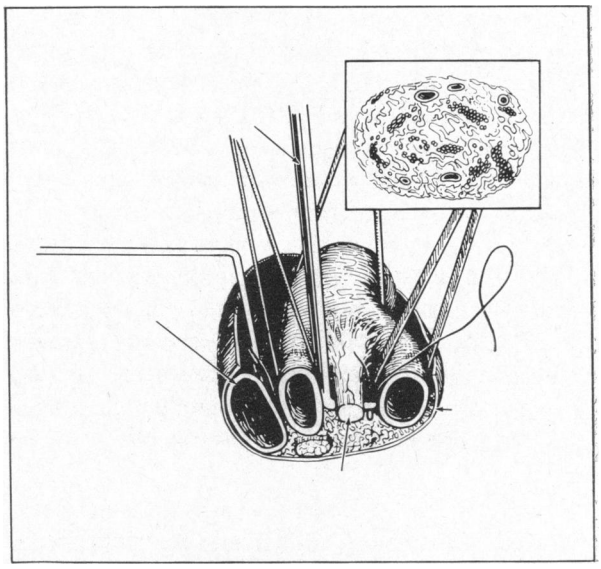

Figure 6.— The carotid sinus nerve which lies in the angle between the internal and external carotid arteries is isolated together with its nutrient vessels before placement of the electrodes about the nerve.

is identified in the bundle of tissue between these two vessels. A heavy silk suture is placed about the nerve, taking care not to isolate the carotid sinus nerve from its nutrient vessels in order to insure permanent viability of the nerve (Figure 6). Also, the terminal branches of the nerve should not be disturbed as they spread out over the carotid bulb, lest the structure be denervated. Dissection of the nerve 1 to 2 cm proximal to the carotid bifurcation insures that these fibers will remain intact. An effort is made to identify the hypo-

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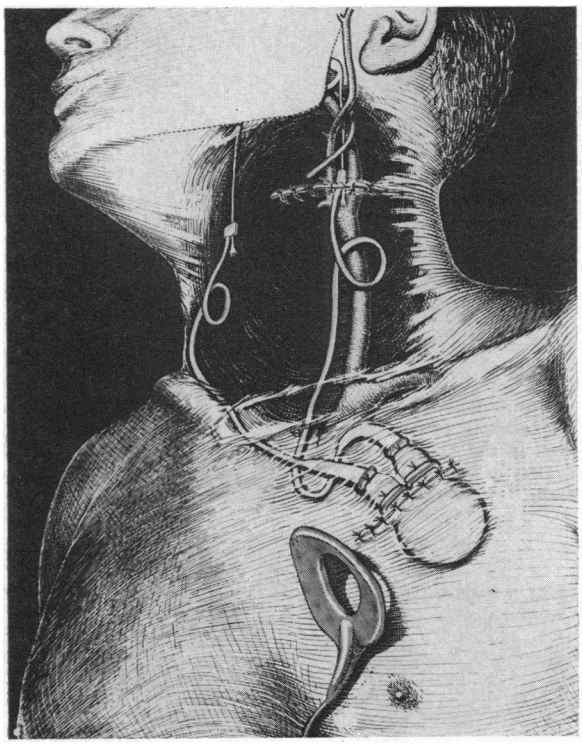

Figure 7.— The receiver is positioned on the anterior chest wall and is connected to the electrode units which are secured about the carotid sinus nerves on each side of the neck.

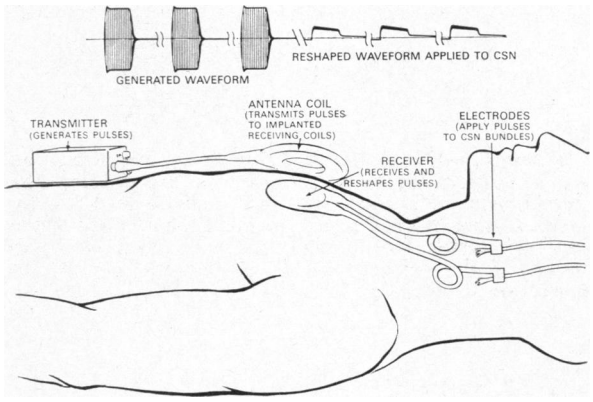

Figure 8.—The shape of the waveform generated by the externally worn transmitter is shown at top of picture. It is generated when the patient places the antenna coil over the receiver and actuates the on-off switch. CSN=carotid sinus nerve.

glossal nerve as it crosses the upper portion of the field. Whenever possible this structure must be protected and undue traction on this nerve assiduously avoided.

Tunnels are then made on each side of the neck with a blunt instrument, connecting the submandibular incisions with the infraclavicular pocket. Both electrodes are passed through the tunnels inside Penrose drains. The silicone jackets of the electrodes are opened and positioned about the carotid sinus nerve before securing the electrodes (Figure 7). The response to a test dose of current is determined to make sure that the unit is functioning satisfactorily, as evidenced by a reduction in systolic blood pressure of 15 to 20 mm of mercury and a slowing of the heart rate by 5 to 10 beats per minute. The silicone jackets around the electrodes are sutured closed and the incisions are also closed, leaving adequate slack loops of the electrodes in the neck to allow for full range of motion of the head and neck postoperatively (Figure 8).

A series of drug solutions is made up before operation and used throughout the procedure to maintain the hemodynamic status of the patient as close to the preoperative level as possible. If the heart rate falls below 55 beats per minute, atropine is administered. Hypotension associated with a normal or fast heart rate is controlled with phenylephrine. If hypotension associated with a slow heart rate develops and is not controlled with atropine, isoproterenol is administered immediately. The systolic pressure is not allowed to fall more than 10 mm of mercury below the patient's usual level. Hypertension is treated with trimethaphan (Arfonad®). Lidocaine hydrochloride is given intravenously for ventricular irritability.

It has been observed that patients complain of a variety of side effects postoperatively, including pain in the operative area and coughing, if the stimulator is used before healing is complete. This is probably due to radiation of the electrical current to branches of the local sensory nerves in the dissected area. Therefore, three to four weeks are allowed to elapse before the unit is activated. During the initial testing period an intensity of stimulation is selected which causes a fall in arterial pressure to a level 15 to 20 mm of mercury below the control, while at the same time relieving the angina.

Two deaths occurred in the immediate postoperative period in our first four patients. These resulted from massive myocardial infarctions; one was due to an episode of bradycardia and hypotension intraoperatively in response to traction on a sensitive carotid sinus nerve. The other occurred in a patient in whom hypoxemia developed following extubation while being moved from the ope-

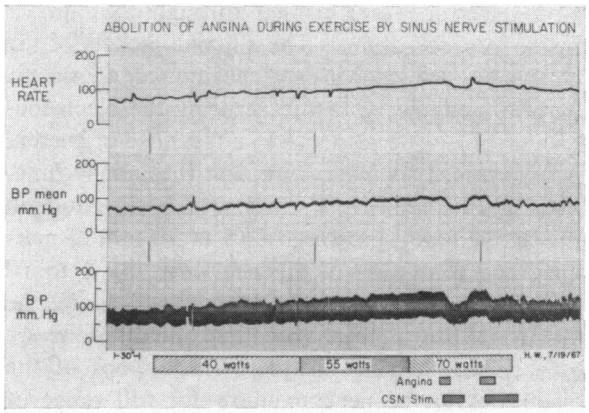

Figure 9.—Representative tracing showing the abolition of angina during exercise by sinus nerve stimulation. The onset of angina occurred during exercise performed at 70 watts. At this point the stimulator was turned on, and heart rate and arterial pressure fell. This was immediately followed by complete cessation of the angina. The stimulator was then turned off. The heart rate and blood pressure rose, and angina recurred. Turning on the stimulator again resulted in decreases in heart rate and arterial pressure and disappearance of the chest pain. (Reproduced by permission from New England Journal of Medicine 277: 1278, 1967.)

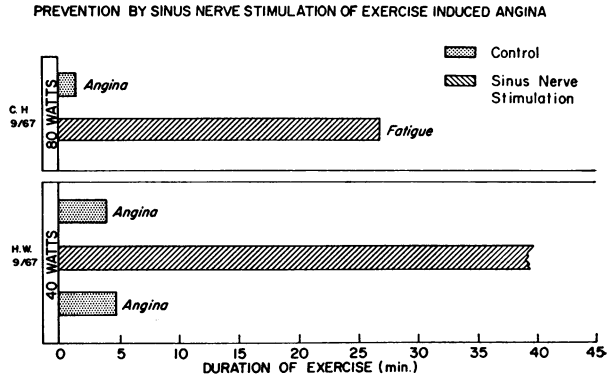

Figure 10.—Effect of carotid sinus nerve stimulation on the capacity of two patients to perform a level of exercise that, in the absence of stimulation, consistently produced angina in less than 5 minutes. (Reproduced by permission from New England Journal of Medicine 277:1278, 1967.)

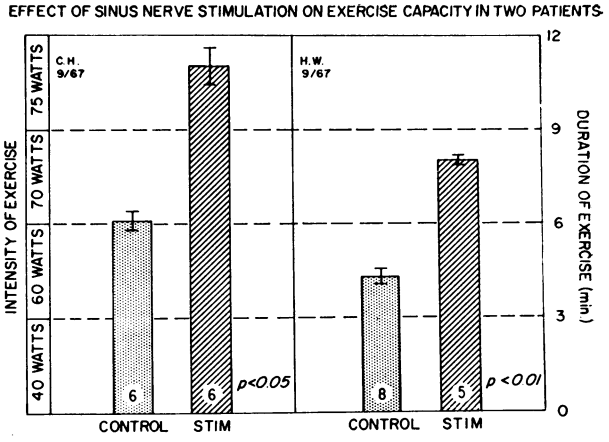

Figure 11.—Effect of carotid sinus nerve stimulation on the intensity of exercise that could be achieved before the development of anginal pain. The exercise load was increased every 3 minutes, and exercise was stopped at the onset of angina. The number of trials is shown at the bottom of the vertical bars. (Reproduced by permission from New England Journal of Medicine 277:1278, 1967.)

rating room to the recovery room. The subsequent 18 patients have all survived the operation. Bilateral hypoglossal nerve paralysis occurred in one patient as a result of undue traction on the nerve, and temporary unilateral paralysis was noted in two others. In all instances there was complete recovery over the ensuing months.

The candidates for insertion of the carotid sinus stimulator represent an especially high risk group of patients even for a relatively simple operation because of their far advanced coronary artery disease. Hence, if the operative mortality is to be kept to a minimum, particular attention must be paid to even the most minute details of intraoperative and postoperative management. Thus, the blood pressure and heart rate must not be allowed to deviate from the normal. Oxygen saturation must be optimally maintained at all times. This can be facilitated by measuring blood gases regularly and leaving the endotrachial tube in place until adequate ventilation is assured.

Results

DR. E. BRAUNWALD: Fifteen of the 20 surviving patients experienced striking symptomatic improvement.[17,18] They reported that they could terminate all or almost all episodes of angina by carotid sinus nerve stimulation. Prophylactic use of the stimulator—that is, activating the device before exertion which would ordinarily produce angina—allows them to perform more strenuous activities without the occurrence of angina and the consumption of nitroglycerine was decidedly reduced (Figures 9, 10, 11). Two patients did not appear to benefit from the stimulator while two experienced only moderate relief of symptoms. One patient was operated upon too recently to allow evaluation. In the majority of patients exhibiting ST-segment depression during exercise, prophylactic activation of the stimulator before exercise caused changes in the ST segment to begin later during the course of exercise, and at any given

time during exercise the ST segment depression was less than without stimulation.[18]

Stimulation of the carotid sinus nerves appears to be superior to nitroglycerin for several reasons. It is more rapid in onset, and since relief of angina is complete within seconds, the patients are not forced to interrupt the activity that precipitated the angina; it is more reliable in consistently aborting each anginal episode and it is not accompanied by some of the undesirable side effects of nitroglycerin, such as headache, a pounding pulse and a feeling of faintness in the upright position. In addition, this method of relieving anginal attacks seems to be preferable to the chronic administration of beta-adrenergic receptor blocking agents since sinus nerve stimulation deprives the heart of sympathetic support only intermittently—that is, at the time angina actually occurs or is likely to occur. However, the combined use of the blocking agents and intermittent sinus stimulation may be more effective in the control of angina than either one alone. Another advantage of sinus nerve stimulation is that because of its predictability and reliability it allows initiation of a program of increasing physical activity in patients with severe coronary artery disease. Such a program may help to induce formation of collateral vessels and thereby alter favorably the natural history of the illness.

Although these results are promising, it must be emphasized that carotid sinus nerve stimulation is not without hazard. Insertion of the electrodes and the receiving unit requires an operation under general anesthesia, a procedure that carries some risk in patients with serious coronary artery disease. However, the development of a plan for the intraoperative and early postoperative management of these patients[15,16] and careful adherence to this plan, should minimize future risks. It should be emphasized that not all patients with angina pectoris are candidates for this method of treatment since most of them experience relief by medical management alone. However, those patients who are severely incapacitated by angina despite optimal medical management should, I believe, be considered for carotid sinus nerve stimulator implantation. Also, it is clear that there is no incompatibility between carotid sinus nerve stimulation and other operative methods for treating angina. Thus, in the event of failure of nerve stimulation, a revascularization procedure or direct bypass of the coronary obstruction could still be carried out later. Also, and in our experience much more commonly, the reverse may be the case and patients who have not benefited from myocardial revascularization are considered for insertion of a carotid sinus nerve stimulator. It is our current policy in patients with intractable angina to consider carotid sinus nerve stimulation first, since it is a simpler procedure, and to hold one of the other surgical procedures in reserve.

As indicated earlier in the conference, manual stimulation of the carotid sinuses frequently abolishes attacks of supraventricular tachycardia. Dr. Sobel will now describe the application of electrical stimulation of the carotid sinus nerves in a patient with this condition.

Use of Stimulation in Supraventricular Tachycardia

DR. BURTON E. SOBEL*: On the basis of observations in patients with angina pectoris and patients with hypertension the efficacy of implanted radiofrequency carotid sinus nerve stimulators in providing a safe means for initiating reflex vagal activity is now established. Electrical stimulation of the carotid sinus nerves offers several advantages over manual stimulation to produce reflex activation of the vagus nerves. The possibility of trauma to the carotid artery is avoided, as is the risk of dislodging a thrombus from the vessel and of interference with cerebral blood flow. Accordingly, we elected to use this approach in a patient with incapacitating, recurrent supraventricular tachycardia resistant to conventional methods of treatment.[19]

The patient was a 69-year-old man with a 21-year history of well documented, recurrent supraventricular tachycardia. Some episodes were clearly paroxysmal atrial tachycardia while others were paroxysmal nodal tachycardia. During the year preceding admission, the bouts of arrhythmia increased in frequency and severity. Attacks lasted for periods of as little as 10 minutes to as much as 17 hours and occurred seven to ten times a week. They were accompanied by diaphoresis and often followed by chest pain. Treatment such as manual pressure on the carotid sinus or administration of intramuscular metaraminol became less effective in terminating attacks during the year preceding admission. A vigorous medical regimen, including large doses of procaine amide and quin-

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idine, and combinations of other agents including digitalis, diphenylhydantoin, atropine, and propranolol were ineffective in preventing attacks. Physical findings at the time of admission were unremarkable, as were the usual laboratory studies. The electrocardiogram showed left axis deviation, parietal block, a wandering atrial pacemaker, and evidence of an old anteroseptal myocardial infarction.

During the first few weeks following implantation of the stimulator several episodes of paroxysmal supraventricular tachycardia occurred and were successfully terminated by activation of the carotid sinus nerve stimulator. However, when hypotension accompanied the arrhythmia, the carotid sinus nerve stimulator was effective only when systemic arterial pressure had been elevated with phenylephrine. The need for additional pharmacologic therapy rapidly diminished and after the second postoperative month activation of the stimulator alone was consistently effective in terminating bouts of supraventricular tachycardia.

More recently we treated a 65-year-old woman with recurrent atrial tachycardia with a carotid sinus stimulator. Although it is too early for definitive evaluation, the initial results are also encouraging; the patient stopped her last episode of tachycardia with the stimulator.

The mechanism responsible for paroxysmal supraventricular tachycardia appears to be either rapid discharge of an ectopic pacemaker or reciprocal beating. In either case, vagal stimulation has a salutary effect by diminishing automaticity or slowing conduction through the atrioventricular junction. Our initial experience with this therapeutic approach has been gratifying and the use of a stimulator avoids the small but definite risk attendant on repetitive manual carotid sinus massage. Since the radiofrequency stimulator can be activated by the patient at will, and since patients are immediately aware of the onset of supraventricular tachycardia, radiofrequency stimulation of the carotid sinus nerves offers obvious advantages in the treatment of this arrhythmia. Other methods of producing reflex vagal stimulation, such as the administration of pressor drugs, may be associated with a cerebrovascular accident, myocardial damage, or pulmonary edema due to transient but pronounced systemic arterial hypertension. However, it must be acknowledged that the long range benefits and hazards of radiofrequency stimulation of the carotid sinus nerves in the treatment of supraventricular tachycardia are yet to be defined.

DR. E. BRAUNWALD: In conclusion, while considerable information concerning the function of the carotid sinus reflex in anesthetized animals has been available, the precise role of this reflex in circulatory control in intact conscious animals and in man had not been clarified. The experiments on conscious dogs and the clinical observations on patients with implanted carotid sinus nerve stimulators described in this conference are providing an increased understanding of this important reflex. It appears that the ability to activate this reflex in patients allows control of intractable angina pectoris and of recurrent supraventricular tachycardia. The early results have been sufficiently encouraging to warrant continued trial of this new mode of therapy.

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